

Research article

Prior Knowledge of Trial Number Influences the Incidence of Plateau at VO₂max

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Abstract

The purpose of this study was to assess the VO₂max plateau response at VO₂max during a series of pre-determined trials. METHODS: Ten male well-trained athletes (age, 23.0 ± 3.2; height, 183.3 ± 5.5 cm; mass 77.5 ± 11.1 Kg; VO₂max 66.5 ± 5.0 ml·kg⁻¹·min⁻¹), but who were VO₂max testing naïve and with prior-knowledge of trial number completed four incremental tests to volitional exhaustion, separated by ~72-h for the determination of VO₂max and gas exchange threshold. Throughout all trials VO₂max was recorded on a breath-by-breath basis using a pre-calibrated metabolic cart, using a plateau criterion of $\Delta \text{VO}_2 \leq 1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ over the final 2 consecutive 30 s sampling periods. A significant difference was observed between trial-1 and trial-4 for plateau incidence ($p = 0.0285$) rising from 20% in trial-1 to a 70% response rate in trial-4. Furthermore a significant difference was observed for VO₂diff (difference between criterion value and ΔVO_2) in trial-1, $1.02 \pm 1.69 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($p = 0.038$), with non-significant differences observed for all other trials, despite a non-significant difference for VO₂max across all trials ($p > 0.05$). Finally, a significant difference was observed for effort perception (RPE) at volitional exhaustion between trial-1 (17.7 ± 1.3) and trial-4 (19.0 ± 1.4) ($p = 0.0052$). These data indicate that prior-knowledge of trial number can influence the manifestation of the VO₂ plateau in a group of well-trained male athletes, thereby suggesting that a form of effort control is established in order to preserve the finite anaerobic capacity.

Key words: Maximal oxygen uptake; effort control; anaerobic capacity; experience.

Introduction

The classical outcome of a maximal oxygen uptake test (VO₂max) is the manifestation of a plateau-like response in VO₂, in spite of a continued increase in exercise intensity, the so called ‘true’ VO₂max. First identified and defined by Hill and Lupton (1923) VO₂max represents the uppermost boundary for aerobic metabolism and reflects the integrated response of the cardiovascular, respiratory and muscular systems to take-up and utilise oxygen. The conventional understanding for the generation of the plateau in VO₂, towards the end of such a test, is that an imbalance ensues between the demand for oxygen at the engaged muscle, as expressed by the arterio-venous oxygen difference (a-vVO₂diff) and the ability of the cardiorespiratory system to supply oxygenated blood to the

muscle to meet the imposed demand, which at sea-level is primarily limited by the cardiac output (\dot{Q}). As volitional exhaustion approaches, both \dot{Q} and a-vVO₂diff exhibit a plateauing response (Calbet et al., 2007) with VO₂ as measured directly at the mouth, also levelling-out. Accordingly the plateau continues to be considered as the primary criteria in establishing VO₂max (Hill and Lupton, 1923; Shephard et al., 1968). However there is an increasing body of evidence which suggests that there are significant variances in reported plateau incidence manifest as a function of athlete ability, ergometer selection, VO₂ sampling rates and exercise protocol (Astorino, 2009; Doherty et al., 2003; Gordon et al., 2012).

A possible contributor to this variance in plateau incidence is the differential ability to recruit type II muscle fibres and hence regulate anaerobic substrate metabolism as recently proposed (Hawkins et al., 2007; Gordon et al., 2011). Thus it has been suggested that the levelling off in VO₂ is dependent on the size of the finite anaerobic capacity, with a significant negative relationship being observed between the ΔVO_2 during the final 60 s of the incremental test and the surrogate measure of anaerobic capacity, maximally accumulated oxygen deficit (MAOD) (Gordon et al., 2011). Further support for these conclusions has come from work showing that when the VO₂max trial was preceded by a bout of prior-priming exercise, in the heavy or severe exercise domains, plateau incidence increased by 50 and 35%, respectively, from a baseline response rate of 50% in the un-primed state (Gordon et al., 2012). The proposed rationale for this response being that such prior-priming spares the finite anaerobic capacity at the onset of exercise by reducing the size of the O₂ deficit.

During closed-loop exercise, such as time-trialling in cycling, it is generally accepted that the participant adopts a pacing strategy in order to optimise performance (Ansley et al., 2004; Foster et al., 2004; Hettinga et al., 2006). Pacing strategies have been attributed to maximising substrate metabolism whilst compensating for the consequences of fatigue (Noakes and St Clair Gibson, 2004; St Clair Gibson et al., 2006). Accordingly it is proposed that exercise intensity is modulated in response to afferent signals from biological and psychological systems, which relay the responses of the exercise challenge to the brain where appropriate efferent, homeostatic-orientated responses are issued. The rationale for these

modulations in pace is to ensure that the finite anaerobic capacity never becomes fully depleted (Foster et al., 2004; Stone et al., 2012). A primary facet of this model is that regulation of effort is the product of an algorithm whereby an individuals' conscious perception of effort (rating of perceived exertion, RPE) is continuously compared to a sub-conscious template which is, in turn derived from previous exposure to the sensations of pain and fatigue and the expectation of the exercise duration (Billaut et al., 2011; Tucker, 2011). In this connection, recent work by (Green et al., 2010) established that pacing is a product of training status with those individuals, who were more experienced and well-trained, showing a greater propensity for adopting a suitable effort-control response during a close-looped exercise than less experienced counterparts.

This situation contrasts with traditional VO_2max tests where the participant is unaware of the end-point thereby creating an open-looped condition. Open-looped exercise poses a potential conflict to the pacing model, as it has been suggested that in order to regulate pace and thereby effort, an endpoint is needed (Mauger and Sculthorpe, 2012). Given that the pacing/effort paradigm is based upon the establishment of a perceptual-based template, the contention is that such a template could be developed purely in response to the sensations of pain and fatigue established during initial experience of these conditions. Hence during a series of repeated trials there would be a regulation of force-output and substrate utilisation through anaerobic pathways in response to the previously established sub-conscious template (Stone et al., 2012). Thus the *a-priori* hypothesis was that in a group of VO_2max testing naïve participants the incidence of plateau would increase across a series of trials and be highest in the final trial due to the development of the perceptual-based template derived from the need to conserve the finite anaerobic capacity in earlier trials. Accordingly the purpose of this study was to examine if prior knowledge of a VO_2max test influences the manifestation of the plateau at VO_2max in a series of subsequent incremental tests to volitional exhaustion in a group of well-trained individuals.

Methods

Participants

Following local institutional ethical approval (Anglia Ruskin University, UK) and having provided written and informed consent a total of 10 male trained cyclists volunteered to participate in the study (age, 23.0 ± 3.2 yrs; height, 1.83 ± 0.06 m; mass 77.5 ± 11.1 Kg). If any participant indicated a contraindication to exercise such as asthma, recent infection, or hypertension, they were excluded from the study. The criterion for classification of trained was a $\text{VO}_2\text{max} \geq 60 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and participation in aerobic endurance training > 3 times per week for > 3 years. An additional key inclusion criterion was imposed, that the participants had not undertaken any form of VO_2max testing prior to this study. Throughout the course of the study the participants were encouraged to maintain their normal daily and training routines, but to refrain from any physical activity in the 24-h period preceding

any laboratory test. All participants were instructed to report to the laboratory fully hydrated and having consumed a balanced meal at least 3-h prior to the test.

Study design

Each participant reported to the laboratory on four separate occasions to undertake an incremental test to exhaustion, with all trials being at the same time of day so as to minimise diurnal variation, with each visit separated by at least 48-h, but no longer than 96-h. Four trials were selected as they would allow for an understanding of the effect of prior-knowledge but would not have a large enough time frame between the first and last trial to be significantly affected by training and de-training responses. During each visit the participants completed an incremental test to exhaustion on an electronically controlled cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands) for the determination of VO_2max and gaseous exchange threshold (GET). The geometry of the cycle ergometer was established for each participant during the first trial and then maintained for all subsequent trials. The participants were not made aware of the primary rationale for the study and were simply informed that the focus of the research was on VO_2max repeatability.

VO₂max protocol: For the determination of both GET and VO_2max the participants undertook an incremental ramp test to volitional exhaustion, from an initial workload of 100 W for 1 min followed by a ramped increase in resistance of $0.42 \text{ W}\cdot\text{s}^{-1}$. For all trials the participants were asked to maintain a constant cadence of 80 rpm and the test was terminated when the cadence decreased by > 5 rpm from that prescribed, or when they reached volitional exhaustion. During each trial, pulmonary gas exchange variables (VO_2 , VCO_2 , VE and RER) were recorded on a breath-by-breath basis using a pre-calibrated metabolic cart (MSX 671; Ferraris Respiratory, Middlesex, UK). Heart rate responses were also recorded, continuously, throughout the course of all trials (Polar 810s, Kempe, Finland), with the data averaged on a 15 s basis. Additionally RPE was ascertained using the 6-20 scale and was collected both pre and immediately upon completion of the exercise challenge. For trial-1 the incremental test was preceded by a self-selected warm-up which was also monitored. To ensure consistency across the remaining trials (2-4) the initial warm-up was standardised for each individual using that adopted for trial-1. Throughout all of the trials verbal encouragement was regulated in accordance with previous work (Andreacci et al., 2002). No verbal encouragement was given until 6 min of the test had elapsed and here it was applied in the form of a chosen phrase such as "you're doing well". Similarly no further encouragement was given until an RER of 1.0 was observed when a reminder to the participant to maintain their selected cadence was provided. Finally no information was provided to the participant regarding their performance for trials 1-3, however upon completion of trial 4 they were given a full debrief as to what they had achieved.

For all trials a VO_2max was confirmed according to previously established methods (Doherty et al., 2003; Lucia et al., 2006) of a ΔVO_2 over the final 2 30 s sam-

pling periods $\leq 1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, which was designated as a plateau response. In the absence of a plateau, a maximal effort was established and a $\text{VO}_{2\text{peak}}$ confirmed according to previously established 'secondary' criteria: $\text{RER} \geq 1.15$, $\Delta\text{RER} \geq 0.4$, peak blood lactate (pBLa) $\geq 8.0 \text{ mM}$, a $\text{RPE} > 19$ (Gordon et al., 2012) and a maximal heart rate (HR_{max}) $205.8 - 0.685(\text{age}) \pm 3 \text{ b}\cdot\text{min}^{-1}$ (Inbar et al., 1994). If neither the primary (plateau), nor secondary criteria were met, the test was deemed a non-maximal effort and discarded. Additionally GET was determined according to the excess CO_2 method (ExCO_2) (Volkov et al., 1975), where ExCO_2 reflects an exercise intensity where the production of CO_2 exceeds that witnessed under steady-state conditions and is expressed as $((\text{VCO}_2^2 / \text{VO}_2) - \text{VCO}_2)$.

Prior to all trials commencing, baseline capillary blood samples were collected for the automated analysis of haemoglobin ($5 \mu\text{l}$) (β -haemoglobin, HemoCue, UK), haematocrit ($10 \mu\text{l}$) and erythrocytes ($10 \mu\text{l}$) (Dr LP20 Miniphotometer, Dr Lange, Germany). Additionally blood lactate samples were recorded both pre and immediately post exercise ($10 \mu\text{l}$) (GM7 Micro-Stat analyser, Analox Instruments, UK). All equipment was calibrated according to the manufacturers' instructions.

Pulmonary gas exchange variables: During all of the incremental trials respiratory volumes and flow were determined with the participant breathing through a low resistance mouthpiece and turbine assembly. Expired gas concentrations (O_2 , CO_2 , N_2 and Ar) were analysed continuously at a rate of $60 \text{ ml}\cdot\text{min}^{-1}$ via a fine-wire capillary line of 2 m and a bore of 0.5 mm connected to the mouthpiece assembly. Using custom metabolic cart software, respiratory volumes and gas concentrations were aligned and processed to obtain respiratory gas exchange variables (VO_2 , VCO_2 , VE , RER). Prior to each trial, the metabolic cart was calibrated in line with manufacturers' specifications and in accordance with previous studies. In accordance with previous studies (Astorino and White 2010; Midgley et al., 2006) the coefficient of variation within our laboratory for athletes of a similar age and training status using the same protocol as adopted for this study is 3.4%.

Statistical analysis

The plateau in VO_2 was calculated along with all of the aforementioned secondary data, means and standard deviations were derived for all variables. Using Levene's test for homogeneity of variance the data was shown to be both normally distributed and homogenous. As this was the case a repeated measures ANOVA was applied to assess the null hypothesis that athlete experience has no influence on the indices of $\text{VO}_{2\text{max}}$, GET and associated sub-maximal responses. To determine the presence, or absence, of a plateau the slope in VO_2 during the final 60 s of the incremental test was determined using least squares regression. Confirmation of plateau manifestation was evaluated using a non-parametric binomial test, where $\Delta \text{VO}_2 \geq 1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} = 0$ (no plateau) and $< 1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} = 1$ (plateau). A binomial test was also used to assess $\text{VO}_{2\text{dif}}$ = the difference between the derived ΔVO_2 and the prescribed ΔVO_2 criterion value for a plat-

eau response of $1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, where $\text{Trial } 1 > \text{Trial } 4 = 1$ and $\text{Trial } 1 < \text{Trial } 4 = 0$.

Plateau incidence was also confirmed using a repeated measures ANOVA which was applied to the regression slope of the VO_2 data to determine if there was any treatment x participant interaction. For the RPE data, a non-parametric Kruskal-Wallis ANOVA was applied. For all statistical analysis the alpha level was set at $p < 0.05$ and all analyses completed using SPSS version 20 (SPSS, Chicago, IL).

Results

Of the 10 participants only 2 met the primary criteria for a plateau in VO_2 at $\text{VO}_{2\text{max}}$ ($< 1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) for trial-1. For trial-2 the response rate was 5 out of 10 which was also repeated for trial-3 while for trial-4 the response rate was 7 out of 10 participants. This indicates a significant difference between trials 1 and 4 ($p = 0.028$) and a trend towards increasing plateau attainment from trial-1 to -4. Although not showing a significant difference, the ΔVO_2 across the final 2 30 s sampling periods demonstrated a distinct negative trend of $1.23 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ between trials-1 and -4. This trend is illustrated in Figure 1, where 9 out of 10 participants exhibited a $\text{VO}_{2\text{dif}}$ in trial-4 that was less than those in trial-1. Here a binomial test revealed a significant difference ($p = 0.02$) in VO_2 between trials-1 and -4. Additionally when the ΔVO_2 response during the final 60 s of the test was directly compared to the plateau criteria of $1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($\text{VO}_{2\text{dif}}$) a significant difference was observed for trial-1, 1.02 ± 1.69 ($p = 0.04$), with non-significant differences revealed for trial-2, 0.17 ± 1.11 , trial-3, 0.18 ± 1.24 and trial-4 of $-0.22 \pm 0.78 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($p > 0.05$). Non-significant differences were observed for all other trial interactions ($p > 0.05$).

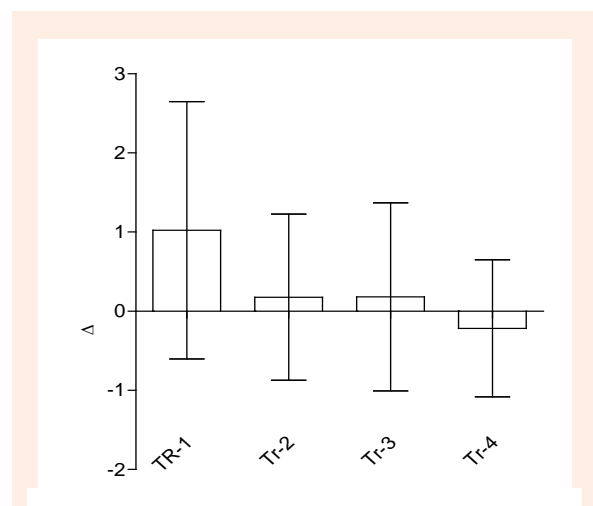


Table 1. Physiological and performance derived responses across the four trials. Data are means (\pm SD).

	Trial 1	Trial 2	Trial 3	Trial 4
VO ₂ max (l·min ⁻¹)	4.98 (.55)	4.97 (.46)	4.94 (.47)	5.12 (.57)
VO ₂ max (ml·kg ⁻¹ ·min ⁻¹)	66.45 (5.02)	66.42 (5.09)	65.95 (5.03)	68.78 (7.05)
Δ VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	1.16 (1.94)	1.29 (1.75)	1.59 (1.27)	.86 (1.33)
VCO ₂ max (l·min ⁻¹)	5.32 (.52)	5.31 (.64)	5.43 (.41)	5.31 (.47)
VCO ₂ max (ml·kg ⁻¹ ·min ⁻¹)	71.16 (5.49)	70.70 (5.79)	72.69 (6.40)	71.42 (6.79)
Δ VCO ₂ (ml·kg ⁻¹ ·min ⁻¹)	2.27 (2.03)	2.46 (1.82)	2.87 (1.25)	1.72 (1.48)
RER _{max}	1.08 (.07)	1.08 (.08)	1.11 (.06)	1.05 (.06)
VE _{max} (l·min ⁻¹)	184.15 (17.22)	185.36 (18.74)	189.82 (20.52)	188.39 (22.81)
GET %VO ₂ max	59.36 (5.38)	59.38 (7.79)	59.73 (6.33)	59.36 (4.35)
HR _{max}	184.2 (10.3)	183.1 (7.8)	181.5 (8.9)	183.5 (10.8)
BLa _{max}	9.80 (2.30)	9.29 (2.75)	8.72 (1.49)	9.56 (2.61)
W _{max}	422.75 (22.39)	425.63 (18.95)	421.85 (14.72)	426.05 (14.93)

Where Δ VO₂max = change in VO₂ over the final two consecutive 30 s sampling periods, Δ VCO₂ = change in VCO₂ during the final two consecutive 30 s sampling periods, RER_{max} = respiratory exchange ratio obtained at VO₂max, VE_{max} = minute ventilation at VO₂max, GET% VO₂max = gas exchange threshold expressed as a % VO₂max, HR_{max} = heart rate recorded at VO₂max, BLa_{max} = blood lactate concentration recorded at the point of volitional exhaustion, W_{max} = power output derived at volitional exhaustion.

trial ($p > 0.05$). The RPE's recorded at the point of test termination across the four trials were 17.7 ± 1.3 , 18.3 ± 1.4 , 18.7 ± 1.1 and 19.0 ± 1.4 for trials -1 to -4 respectively. These data revealed a highly significant difference for effort perception between trial-1 and trial-4 ($p = 0.005$). Additionally when considering the RPE within trials it was shown to be significantly, but negatively correlated against VO₂dif for trial-1, $r = -0.658$ ($p = 0.04$) and positively correlated for trial-4, $r = 0.654$ ($p = 0.04$).

The haematological responses obtained prior to trial-1 were 144.3 ± 13.8 gdl⁻¹ (Hb), 44.7 ± 5.2 % (Hct) and (Ery) 4.81 ± 0.56 (mio· μ l⁻¹), with no significant changes in any of these variables across the 4 trials ($p > 0.05$). All performance and physiologically derived responses to the four incremental test trials are presented in Table 1, which shows that there were for the majority of indices no-significant differences across trial.

Discussion

The purpose of this study was to explore whether during a series of repeated, traditionally orientated VO₂max trials, a controlled-effort was employed. An *a-priori* hypothesis was established based on the predication of pacing being employed as a response to the need to modulate substrate metabolism across trials, to ensure that the finite anaerobic capacity never becomes fully depleted (Foster et al., 2004; Tucker, 2011). Since the plateau in VO₂ at VO₂max is a function of the same finite anaerobic capacity (Hawkins et al., 2007; Gordon et al., 2011), it was further hypothesised that plateau incidence would increase with repeated exercise trials. The reported findings support this hypothesis showing an increased incidence of plateau response between trial-1 and trial-4, coupled with a significant difference in VO₂dif between trial-1 and trial-4.

These differences were manifest despite no other changes in measured responses, including exercise time to exhaustion, VO₂max or GET.

During an incremental test to exhaustion, using a similar protocol to that adopted for the current study, it has previously been demonstrated that there was a significant decrease in the finite high energy phosphate capacity (Green and Patla, 1992). Indeed Green et al., (1992) indicated that upon arrival at VO₂max and hence volitional exhaustion, the PCr concentration decreased by 86% in approximate proportion to the increase in free Pi. As a consequence of the increasing exercise intensity and associated decline in the PCr concentration, there was a notable decrease in the intramuscular glycogen concentration, coupled with an increase in muscle lactate and IMP, resulting in a significant decrease in muscle pH (Bertuzzi et al., 2013; Cooke et al., 1988; Greem and Patla, 1992). Previous work suggests that effort-control strategies are adopted through a conscious and/or subconscious desire to limit the onset of premature fatigue through a modulation of work over the desired task duration (Billaut et al., 2011; Tucker 2011). By integrating this modulation of work to the metabolic changes associated with fatigue, (H⁺, pH, decreasing muscle glycogen, PCr etc.) skeletal muscle mass recruitment can be regulated to match mechanical output against performance (Billaut et al., 2011; Foster et al., 2004; St Clair Gibson et al., 2006). Such a strategy would be established at the onset of the exercise challenge, based upon previous associated sensations of pain and fatigue (Billaut et al., 2011; St Clair Gibson et al., 2006), current physiological (substrate availability, metabolic by-products etc.) and psychological state (motivation, arousal etc.), together with a perceptually regulated

Table 2. Time orientated responses across the four trials. Data are means (\pm SD).

	Trial 1	Trial 2	Trial 3	Trial 4
Total (s)	834.6 (53.7)	841.5 (45.5)	832.4 (35.3)	842.5 (35.8)
VO ₂ max-arr (s)	813.6 (52.2)	817.5 (38.7)	811.4 (33.3)	818.5 (32.9)
VO ₂ max-Tlim (s)	21.0 (12.6)	24.0 (14.5)	21.0 (12.6)	24.0 (14.5)
GET (s)	479.0 (52.2)	484.9 (65.3)	485.2 (58.9)	481.8 (44.4)
GET – VO ₂ max(s)	337.2 (51.0)	332.6 (71.3)	326.3 (49.7)	341.5 (34.1)

Where Total = total exercise time from trial onset to volitional exhaustion, $\text{VO}_{2\text{max-arr}}$ = time taken from exercise onset to the onset of $\text{VO}_{2\text{max}}$, $\text{VO}_{2\text{max-Tlim}}$ = time from $\text{VO}_{2\text{max}}$ arrival to volitional exhaustion, GET = time taken from exercise onset to the gas exchange threshold and $\text{GET} - \text{VO}_{2\text{max}}$ = time taken from gas exchange threshold to the point of volitional exhaustion.

response to the perceived exertion (Tucker, 2011).

However, in the present study the participants were $\text{VO}_{2\text{max}}$ testing naïve so for trial-1 had no previously established perceptual template against which to modulate their effort, but were fully aware of the total study duration (4-trials). Accordingly for the initial exercise challenge (trial-1), participants had no perception of the end-point against which work could be modulated, hence rendering the feedback from the engaged muscles redundant in this context. In this connection it is interesting that trial-1 exercise time was, on average, 8 s shorter than that of trial-4 (longest), supporting the contention that for trial-4 there was a perceptual template against which work could be modulated.

Of course the participants in the present study were not totally naïve to the nature of the protocol employed as they were both well-trained and aware of the total number of trials which they needed to perform. The latter factor is significant to an effort-control paradigm, which projects that an exercise end-point is a fundamental component to the perceptual template in regards to the allocation of both physiological and psychological assets (Billaut et al 2011; St Clair Gibson et al., 2006). The paradigm based upon the notion of effort modulation which is a function of previously established homeostatic or reference sensations (Damasio et al., 2000; 2001), which becomes a permanent set-point against which all subsequent exposures to the same activity are compared. The contention being that where the exposure is not immediate the perceptual regulation of effort ensues.

When an exercise end-point is not known (number of trials) there is a down regulation of muscle activity in order to reduce the metabolic cost and thereby spare the finite anaerobic capacity (Billaut et al., 2011). It is contended that the plateau variance, observed in the present study, is a function of the prior knowledge of the number of trials to be completed, so a maximal effort could be applied in the final (fourth) trial. Trial-1 (two) and -3 both showed an identical but increased (50%) plateau incidence and by association a greater reliance on the finite anaerobic capacity than for trial-1, suggesting a rationing of the finite anaerobic resources as a consequence of prior knowledge from trial-1 but a recognition that the final trial (4th) beyond which there was no requirement for sparing the anaerobic reserve had not been reached.

Recent work (Gordon et al., 2013), studying the effects of blood donation, emphasise the process of sparing the anaerobic capacity during incremental testing. Here, despite a reduction in blood volume of $\sim 450 \text{ mm}^3$ and associated decrease in O_2 carrying capacity of $\sim 9\%$, there was no change in plateau incidence, suggesting that during an incremental test to exhaustion the finite anaerobic capacity is still not fully depleted. So although in the present study the participants could not modulate mechanical force output the data would suggest that they adopted a metabolically orientated control of effort, in response to prior knowledge of both trial number and exposure to the sensations of pain and fatigue.

The training status of the participants in the study is also of importance to the outcomes observed. Previous work, (Green et al., 2010) highlights that pacing is a function of training volume and experience. All the cyclists in the present study were well-trained endurance athletes, ($\text{VO}_{2\text{max}}$: $66.5 \pm 5.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and training history of >3 years). Hence whilst the detailed training history of the participants was not known, it is accepted that in order to enhance endurance capability the athlete would need to undertake both low intensity and interval-based training (Billat, 2001; Seiler et al., 2006). Since the latter would typically be in excess of the GET the athlete would be subject to the sensations of pain and fatigue, similar to those experienced during an incremental exercise test.

In a recent study Scharhag-Rosenberger et al., (2014) demonstrated that with just 1.5-h recovery between $\text{VO}_{2\text{max}}$ trials, four incremental tests could be completed with no-significant effect on either $\text{VO}_{2\text{max}}$ or W_{max} . This was in a group of trained endurance athletes, displaying similar physical characteristics to those completing the present study. The lack of change in $\text{VO}_{2\text{max}}$ reported by Scharhag-Rosenberger et al. (2014) is in agreement with that shown in the present study. These findings along with others (Hawkins et al., 2007; Wagner, 2000) suggest that although $\text{VO}_{2\text{max}}$ is primarily limited by cardiac output and O_2 extraction/utilisation at the muscle, the plateau is independent of these responses. Indeed recent works (Calbert et al., 2007) suggest that \dot{Q}_{max} is attained at $\sim 86\% \text{ W}_{\text{max}}$ as a function of a levelling off in stroke volume (SV) at $\sim 64\% \text{ W}_{\text{max}}$. These findings suggest that the maintenance of force generation in response to the continual increase in exercise intensity is a consequence of the reliance on the finite reserves of the high energy phosphates and intramuscular glycogen. Although the relevance of the $\text{VO}_{2\text{max}}$ plateau in establishing a maximal effort has been challenged (Alpert, 1992; Noakes, 2008) its significance should not be under-estimated. For as Hill and Lupton, (1923) first projected the existence of the plateau is central to the notion of a maximal rate of oxygen uptake and conforms to the concept of $\text{VO}_{2\text{max}}$ being dependent on \dot{Q}_{max} . Of note is that in this population group of $\text{VO}_{2\text{max}}$ naïve participants the plateau response rate was lower than reported values in the literature for athletes of equitable fitness (Astorino 2009; Doherty et al., 2003; Gordon et al., 2011). Given that the VO_2 plateau is considered the primary criterion in determination of a maximal effort these findings lend support to the need for such approaches as a verification trial, particularly when the participant is naïve to the exercise challenge. Accordingly debate continues as to the variance in plateau incidence with potential contributors being ergometer type (Gordon et al., 2012), protocol (Kon-Yoon et al., 2007), sampling and analysis methods (Astorino, 2009; Robergs et al., 2010) and population group (Doherty et al., 2003; Lucia et al., 2006). However in order for the debate to be framed and the generation of a series of industry recognised guidelines there needs to be recognition of what the plateau is and represents.

Conclusion

This study has demonstrated that in a group of well-trained male endurance cyclists, a closed-loop condition is established with prior knowledge of trial number which triggers the sparing of the finite anaerobic capacity when exposed to the sensations of pain and fatigue which are evident during such trials. It is proposed that by establishing the closed-loop condition prior to commencement of data collection that a metabolically orientated control of effort ensues which prevents both a depletion of the anaerobic energy reserves and resultant prolonged exposure to the sensations of pain and fatigue. Future work should address whether plateau manifestation shows a similar response pattern in both un-trained individuals who have not had significant exposure to the sensations of exercise-induced pain and fatigue, or in female participants as the majority of research to date focuses on responses in male participants.

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Key points

- In well-trained athletes the incidence of plateau at $\text{VO}_{2\text{max}}$ increases in conjunction with an increase in trial number and the associated sensations of pain and fatigue.
- By informing the participant of the number of trials to be completed a closed-loop condition is developed whereby effort in all trials is compared to a perceptually developed template.
- Closed-loop condition leads to a sparing of the finite anaerobic capacity during incremental tests when the number of trials to be completed is known.

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